Foal Medicine I: Common Problems of Neonatal Foals

Phoebe A. Smith DVM DACVIM
Riviera Equine Internal Medicine & Consulting
smith@rivieraequine.com

Abstract

A good clinician handles common conditions uncommonly well. Common presenting problems in the neonatal period include the floppy/ADR (Ain't Doin' Right: veterinary slang for non-specific concerns of illness) foal, the colicky foal, and the lame foal. This presentation will focus on common causes of these presenting problems. Drug dosages are listed at the end in Table 1.

Primary presenting problem: ADR/floppy foal

The underlying cause of lethargy, malaise or flaccid recumbency in a neonatal foal may be difficult to determine on physical exam alone. A wide range of conditions can result in the presentation of a lethargic, inappetent, or recumbent foal. Primary causes include hypoglycemia, neonatal maladjustment syndrome (NMS), and sepsis. Less common but worth considering as differential diagnoses are uroabdomen and neonatal isoerythrolysis.

Hypoglycemia

The neonatal foal is born in a hypoglycemic state (25 to 45 mg/dL). Glycogen stores in the liver undergo gluconeogenesis to support the foal's blood glucose until it receives its first enteral feeding. Because foals have small reserves of glycogen, blood glucose levels will decrease further between two to four hours postpartum without feeding.[1] This is the reason foals are

expected to stand and nurse within two hours of parturition. A foal with difficulty standing, latching on and/or swallowing will become recumbent in the absence of a glucose source.

Low blood glucose in sick neonatal foals is associated with sepsis, positive blood culture results, and systemic inflammatory response syndrome (SIRS). Both hypoglycemia (<50mg/dL) and hyperglycemia (>180mg/dL) at hospital admission have been associated with non-survival.[2]

Treatment: enteral feeding (100-200mL milk initially) or intravenous dextrose (2.5-5% dextrose) administered as 500mL-1 liter bolus in isotonic fluids initially, then recheck blood glucose

Neonatal maladjustment syndrome

Neonatal maladjustment syndrome, also known as neonatal encephalopathy, hypoxic ischemic encephalopathy, perinatal asphyxia, or dummy foal syndrome is the most common neurological condition of neonatal foals.[3] The various names describe proposed etiologies, but a definitive cause has not been identified to date. Research investigating the role of progestogen neurosteroids in these foals is ongoing.[4] Generalized weakness, decreased affinity for the dam and poor to absent suckle reflex are among the most common clinical signs observed with this syndrome. Blindness, seizure activity and obtundation are less common presenting problems. Clinical signs can either be present at birth or develop in the first 48 hours of life and antemortem diagnosis is typically made by exclusion of infectious or congenital disease.

Treatment: supportive treatment for the clinical signs and any comorbid diseases. Broad spectrum antimicrobial prophylaxis, nutritional and fluid support, careful monitoring of urine production, respiratory pattern, electrolyte and acid-base status. Intensive nursing care required

for recumbent foals to prevent decubital ulcers, self-induced trauma, etc. Additional therapies that may aid in support include thiamine, mannitol, Vitamin C, Vitamin E, and caffeine.

Sepsis

Despite advances in neonatal medicine in recent decades, sepsis remains a leading cause of morbidity and mortality.[5] Partial or complete failure of passive transfer significantly increases the risk of sepsis in neonates. However, sepsis can develop in foals with adequate transfer of passive immunity.[6] Simply stated, sepsis is an overwhelming inflammatory response to microbial invasion via the gastrointestinal tract, umbilicus, respiratory tract, genitourinary tract, or disruption in skin or mucosal barriers. Clinical signs specific to the organ of microbial invasion may be evident in the floppy foal, or signs may be non-specific and limited to malaise and poor suckling. While a positive blood culture provides definitive diagnosis, low sensitivity and time required (48+ hours) for results reporting limit the utility of this tool as a rapid diagnostic. Sepsis scores are used by many clinicians and institutions to aid in the diagnosis, while astute clinical observation remains a valuable means of rapid sepsis diagnosis.[7] The author utilizes the following criteria for rapid diagnosis of sepsis:

Sepsis if two or more of the following are present:

- hypothermia or hyperthermia
- tachycardia
- tachypnea
- leukopenia, leukocytosis, or 10% band neutrophils

Treatment: early initiation of broad-spectrum antimicrobials and excellent supportive and nursing care. In some cases, intensive care, including cardiovascular and respiratory support, and parenteral nutrition is required for successful outcomes.

Primary presenting problem: colicky foal

Causes for colic in the neonatal foal are wide-ranging, as in the adult horse. Among the most common causes in newborn foals are meconium impaction and enterocolitis.

Meconium impaction

Meconium impaction is reported to be the most common cause of colic in neonatal foals, occurring more frequently in colts than fillies. This predisposition is presumed to be due to a narrower pelvic inlet in colts.[7] Meconium is composed of materials ingested in utero: intestinal epithelial cells, mucus, amniotic fluid, bile, and water. Evacuation of meconium begins within three hours of birth in the normal foal and is followed by passage of yellow pasty, digested milk feces. Meconium impaction or retention occurs when attempts to defecate are unsuccessful or incomplete evacuation of meconium occurs.

Additional factors suggested to predispose foals to retention of meconium include maternal malnutrition, delayed colostral intake with loss of its laxative effect, prematurity, dystocia, and dehydration.[8] Most impactions occur in the rectum and/or small colon at the pelvic inlet, but can extend to the dorsal or transverse colon. Discomfort is initially exhibited by decreased nursing, tail flagging/swishing, and restlessness. As discomfort advances, abdominal distension, frequent posturing to defecate, rolling, and lying in dorsal recumbency are seen. Diagnosis is based on clinical signs and detection of firm stool in the rectum on digital palpation, abdominal distension with lack of fecal production, and/or sonographic or radiographic visualization of fecal masses in

the colon. Occasionally, milk feces can be passed around a high meconium impaction, making observation of milk feces alone a poor rule out for the diagnosis of meconium impaction.

Treatment: Simple meconium impactions resolve with one or more enemas, such as commercial phosphate enemas, soapy warm water enemas, glycerin, or mineral oil enemas. Because phosphate containing enemas can result in hyperphosphatemia, these should be used judiciously. Repeated enemas of any type often result in rectal mucosal irritation and swelling. Additional therapy recommended for impactions that do not resolve with simple enema administration include pain control, IV fluids, and mineral oil by nasogastric tube. When first line therapies do not resolve a meconium impaction acetylcysteine retention enema may be administered. Those impactions which are not resolved medically require surgical intervention.

Enterocolitis

Initially, an affected foal may be off milk and lethargic, progressing to colic and diarrhea within hours. Enterocolitis in the neonatal foal commonly results from Clostridial infection, asphyxia-associated hypoxic injury, or necrotizing gastrointestinal (GI) disease. On initial exam of the foal with enterocolitis lethargy, muddy, dark or bright mucous membranes, cool extremities, colic and abdominal distension may precede the development of diarrhea. Ultrasound commonly reveals ileus with flaccid or taut distension of the small bowel. Reflux may be obtained via nasogastric intubation.

Clostridial enterocolitis in foals can occur without predisposing risk factors, in contrast to the disease in adult horses. *Clostridium perfringens* and *C difficile* are the most common clostridial agents involved in neonatal enterocolitis, both occurring sporadically as well as in farm outbreaks. Although the clostridial spores are virtually impossible to eradicate, strict isolation

measures should be utilized when these bacteria are suspected or confirmed. A combination of fecal cultures and toxin assays is recommended to diagnose Clostridial enterocolitis.

Treatment: Metronidazole is appropriate directed therapy, with broad spectrum antimicrobials also recommended to protect against sepsis. Intensive supportive care is often required to correct imbalances of fluid, acid-base, and electrolytes.

Asphyxia-associated enterocolitis is believed to result from ischemic injury to the GI tract occurring in utero or during parturition. Additional organs may show evidence of dysfunction in this case. Diagnosis is based on a history of plausible asphyxia (dystocia, c-section, umbilical cord abnormality) and clinical signs consistent with ileus and intolerance to feeding (colic after nursing/feeding).

Treatment: Conservative enteral feeding via indwelling nasogastric tube, intravenous fluid therapy and broad-spectrum antimicrobials. In some cases, prokinetic drugs are required, in addition to supportive care for all affected organs.

Necrotizing enterocolitis is the most serious of these conditions, believed to result from mucosal injury and maturation abnormalities. The presenting clinical signs and management are similar to those for asphyxia-associated enterocolitis. Typically, necrotizing enterocolitis cases progress very rapidly and require extremely intensive care. In human infants, pneumatosis intestinalis (gas within the mucosal and submucosal layers of the bowel wall) is a prominent feature of the disease.

Treatment: Very limited enteral feeding (trophic only), broad spectrum antimicrobials, parenteral nutrition, vasopressor, fluid, and electrolyte support is required in these cases.[9,10]

Primary presenting problem: lame foal

Lameness in the neonatal foal is considered infectious in origin until proven otherwise in the authors practice. Non-infectious causes of lameness do occur, including traumatic injury, developmental musculoskeletal disease, and inflammatory response to a systemic infection. However, because early recognition and treatment are critical to positive outcomes in infectious lameness, the clinician must rule infection in or out rapidly. Degree of lameness in foals ranges from subtle to non-weight bearing. Observation of the foal on solid, level footing at the walk allows for characterization of the lameness. Additional insight may be gained by allowing the foal to trot alongside the dam in some cases. Palpation of the limbs, joints, and soft tissues is useful as with an adult horse lameness exam.

Non-infectious lameness

Foals sustain injury resulting in lameness at a surprisingly low rate considering the fragility of their apendicular skeletons. Traumatic injury from another horse (kick, stepped on), impact with a stationary object when being chased, and overuse injuries are common causes of non-infectious lameness in the foal. Fractures of the long bones, physes, cuboidal bones, proximal sesamoid bones, pelvis and distal phalanx are observed most often. Pelvic fractures can occur as a result of a foal flipping over or rearing and sitting, while cranial and cervical fractures occur when running foals collide with a stationary object. Cuboidal bone fractures most commonly develop secondary to malformation or crushing in cases of incomplete ossification. Radiography, ultrasonography, and occasionally CT or MRI are used to definitively diagnose these injuries. Developmental orthopedic abnormalities (flexural and angular limb deformities) may cause lameness, though less commonly than injury.

Treatment: specific to the injury or abnormally developed anatomy

Infectious lameness

Infectious lameness occurs commonly in foals. Bacteria gain entry to the bloodstream through the foal's umbilicus, respiratory tract or gastrointestinal tract. Less commonly, external trauma results in direct penetration and subsequent infection of a synovial structure. Hematogenous dissemination of bacteria allows localization into metaphyseal, physeal, or epiphyseal cartilage. Owing to the relatively sluggish blood flow and vascular stasis of nutrient vessels at the cartilage interface, bacteria readily proliferate and colonize. Failure of passive transfer is not a requisite feature of the development of a septic lameness. In foals exhibiting relatively normal behavior, recognition of septic arthritis or osteomyelitis is not difficult. An observant client may first notice joint swelling or lameness. The clinician may recognize heat, edema, or joint effusion on careful palpation of the lame limb. Lameness develops approximately 8-24 hours following colonization of the joint with bacteria.[11] Osteomyelitis-induced lameness is often mild to moderate in severity until the lesion becomes very aggressive. In contrast, septic joint(s) often cause more severe lameness. Recognition of an infected joint or bone in a sick foal may be delayed due to weakness or recumbency. Increased temperature and WBC count are inconsistent and tend to relate more closely to systemic illness. Thorough evaluation of the musculoskeletal system is an important part of the physical exam in a septic foal and should be conducted at least daily. In the recumbent foal, heat, periarticular edema, or pain with palpation or passive motion are early signs of a septic process. Neonates will commonly have multiple joints affected while foals greater than 4 weeks of age are more likely to have 1 joint affected. Any joint can become septic but the larger joints, including the stifle, hock, carpus, elbow and fetlocks are most commonly involved in a septic process in the foal.

Diagnosis of infectious lameness is confirmed with varying combinations of diagnostic imaging (radiography, CT, MRI), increased synovial fluid white blood cell count (>30,000 cells/μL), synovial fluid hyperproteinemia (>3.5 g/dL), increased synovial fluid serum amyloid A (SAA), and synovial fluid culture. Synovial fluid sampling is critical to the diagnosis of a septic joint. Radiographic changes may not initially be present; however, serial radiographic examination should be performed because radiographic changes will often follow clinical signs by up to three weeks. Ultrasonography is beneficial in identifying septic regions of the proximal limb as well as intraarticular fibrin or gas. Additionally, ultrasound can identify abscessation of soft tissues adjacent to septic bone or physis.

Treatment: Repeated local lavage with or without arthroscopic debridement, local and systemic antimicrobials. Regional limb perfusion with antimicrobials is often useful as an adjunctive therapy. Intraosseus antimicrobials are used for cortical bone infections with or without debridement. Hyperbaric oxygen therapy, continuous release antimicrobial therapy and sustained release antimicrobial gels are available adjunctive therapies.

Summary

Familiarity with common causes of lethargy, colic and lameness in the neonatal foal will allow the clinician and his/her team to prepare for the evaluation, time and diagnostics necessary to confirm a diagnosis. Diagnosis begins with careful physical examination, coupled with labwork to determine a presumptive diagnosis as rapidly as possible.

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